Section of Anæsthetics

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Hyperbaric Oxygen

Anæsthesia in a High Pressure Chamber

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The principle underlying the use of oxygen in a high pressure chamber is based on Henry's law, applied to blood: the amount of oxygen which dissolves in plasma is directly proportional to the partial pressure of oxygen in the alveoli.

If room air is inhaled at normal atmospheric pressure, then the partial pressure of oxygen in the alveoli is 100 mmHg. At this tension the arterial oxygen saturation of hæmoglobin is 96% and $100/760 \times 0.023 = 0.003$ ml O_2 or 0.3 vols % oxygen are dissolved physically in plasma (solubility of oxygen in plasma is 0.023 at 38°C and 760 mmHg). If pure oxygen is inhaled the hæmoglobin becomes 100% saturated, holds 20.1 volumes oxygen % and cannot take up any further oxygen. The plasma, however, can take up more oxygen in physical solution. The oxygen tension in the alveoli, after nitrogen has been eliminated, will become $760 - 40 - 47$ mmHg, where alveolar carbon dioxide tension is 40 mmHg and tension of saturated water vapour is 47 mmHg. If 2.3 ml oxygen dissolve in 100 ml plasma, at an oxygen tension of 760 mmHg then, at an alveolar tension of $760 - 40 - 47$ mmHg, $2.3 \times (760 - 40 - 47)/760 = 2.1$ vols % O_2 will dissolve in plasma.

If pure oxygen is inhaled at 3 atmospheres absolute (ATA) then $2.3 \times [3 \times 760 - (40 + 47)]/760 = 6.6$ vols % oxygen will dissolve in plasma in simple physical solution.

The calculated maximum alveolar oxygen tension at 3 ATA would be $3 \times 760 - (40 + 47) = 2,280 - 87 = 2,193$ mmHg with a whole blood oxygen content of 26.7 vols %. The fact that the oxygen tensions which are actually measured in arterial blood are lower than these theoretical calculations can be due to various factors – anatomical and physiological shunts, diffusion factors and shunting in the lungs – which are discussed by Dr McDowall (p 325).

With the body at rest in air at normal pressure, the 19 vols oxygen % in the arterial blood is reduced to 13 vols % in the mixed venous blood. Tissue oxygen requirements are thus about 6 vols % at a normal cardiac output. If a level of 6 vols % oxygen in physical solution could be achieved, then the transport of oxygen from the lungs to the tissues could be provided for by the plasma alone. Reduction of oxyhæmoglobin and even the presence of hæmoglobin itself would no longer be necessary. This state of affairs could be achieved theoretically if pure oxygen were inspired at 3 atmospheres absolute (Haldane & Priestley 1935, Boerema *et al.* 1956). This increase of oxygen in physical solution occurs not only in the plasma but also throughout the whole body, including interstitial fluid and cells. Boerema's term for this was hyperbaric oxygen drenching (HOD).

From animal experiments (Boerema *et al.* 1956, Boerema *et al.* 1959, Meyne, Vermeulen-Cranch *et al.* 1962, Meyne, Keuskamp *et al.* 1962) we have concluded that the time gained under hyperbaric oxygen at 3 atmospheres absolute, compared with other methods now available for intracardiac surgery but not available at the time of the experiments, is mostly too short unless hyperbaric oxygen is combined with hypothermia. This is supported by the results of experiments in which the central retinal artery is compressed by exerting pressure on the eyeball, until anoxic blackout of vision occurs. Vision is blacked out after eight seconds under normal atmospheric conditions; after twenty seconds,

breathing oxygen at 1 atmosphere absolute; after one minute, breathing oxygen at 3 atmospheres absolute. Oxygen 'storage' in the brain is minimal.

Hyperbaric oxygen does offer two advantages during complete circulatory arrest: the storage of oxygen in the tissues before the arrest, and, more important, the availability of blood with a high oxygen tension when circulation restarts, resulting in a more rapid recovery to normal biochemical values, EEG and ECG and in spontaneous defibrillation during massage, compared with events under normal pressure. Thus short intervals of inflow occlusion in hypothermic patients requiring open aortic or pulmonic valvulotomy are possible and safer under hyperbaric oxygen. It is our opinion, however, that hyperbaric oxygen would be of greatest advantage in conditions where at least some circulation is present.

Increasing the oxygen tension in plasma could also be of importance in conditions where the circulation to the tissues is inadequate: hæmorrhagic shock, controlled hypotension, aortic occlusion, carotid artery surgery, vascular trauma, gas gangrene, skin grafts; when right-to-left shunts prevent adequate oxygenation of hæmoglobin; when the oxygen-carrying capacity of blood is reduced as in carbon monoxide poisoning; possibly in the early stages of tissue œdema following tissue damage (after cardiac arrest, after severe carbon monoxide poisoning); and very definitely in the treatment of anaerobic infections. Small children with tetralogy of Fallot have, under hyperbaric oxygen, been submitted to palliative Pott's operation with far less risk, because their tissues are far better provided with oxygen than normally (Boerema *et al*. 1962).

The problem of providing anæsthesia for experimental animals and patients was not the least of the many that arose. Churchill-Davidson *et al*. (1955) described an anæsthetic technique for patients irradiated for carcinoma while breathing hyperbaric oxygen. However, these patients were alone in a small hyperbaric chamber, unaccompanied by an anæsthetist and no surgical procedure was performed. Our problem was rather different. The surgeons and anæsthetists required for a surgical procedure would have to accompany the patient and themselves breathe air at 3 ATA. The patient would have to be anæsthetized and breathe pure oxygen at 3 ATA at least part of the time.

In order to facilitate further progress, a large high-pressure operating chamber was built in the hospital grounds near the surgical clinic in 1959 and first used in August 1960. The entrance to the operating theatre itself is by means of a lock. Since there are two doors between the lock and the operating chamber, it is possible to compress the lock separately and the latter is sometimes also used simultaneously for treatments. The operating theatre is $7 \times 4 \cdot 4$ meters, and has five viewing windows. There is a small lock for passing instruments into the theatre and another into the main lock. The chamber was tested to a pressure of $5 \cdot 6$ ATA. Once the desired working pressure has been reached, one of the two compressors is switched off and the other used for ventilating the chamber at a rate of 4 cu.m/minute. In order to prevent excessive rise of air temperature during compression or excessive fall during decompression, cooling and heating of the filtered inflowing air can be achieved. Operation of the compressors is carried out entirely outside the hyperbaric chamber and, in order to eliminate errors, is never entrusted to less than two people. Members of the medical team inside are, for reasons of safety, unable to control the decompression themselves.

With oxygen at 3 ATA in the airway of the patient, it is not permissible to use any inflammable anæsthetic mixtures inside the chamber. Particular care was taken to earth all equipment and prevent static charges in order to minimize the risk of fire. There is space for a small anæsthesia table with oxygen and air flow meters, a simple gas humidifier and halothane vaporizer to be used inside the chamber. Cylinders are kept outside. Improved monitoring has become possible since specially constructed apertures were incorporated to carry cables for ECG and EEG. Recording takes place outside the chamber by means of an 8-channel Schwarzer recording monitor. A 2-channel oscilloscope for ECG and EEG attached outside one of the viewing windows in the theatre wall enables the anæsthetist inside the chamber to have continuous visual evidence of the state of the heart and brain. A ventilator working on compressed air, such as the Bird or Bennett, can be used provided the pressure of the gas available for driving it can be supplied at $3\frac{1}{2}$ atmospheres above the pressure in the chamber during the period of compression. The capnograph infra-red analyser has proved a useful continuous monitor of the percentage alveolar CO_2 under hyperbaric anæsthesia.

Blood analyses are carried out in the chamber using Astrup equipment for pH, standard bicarbonate and arterial pCO_2. Blood samples for lactic acid and potassium taken under pressure are passed out via the small lock, which is also

used for passing intruments, blood bottles, &c. Oxygen tensions are measured inside the chamber by means of a Gleichmann-Lubbers electrode (Clark system) slightly modified for use under hyperbaric conditions. Blood loss is estimated by weighing and measuring bottles. A defibrillator can be used in the chamber.

Hypothermia can be produced by means of a water bath, but the Amsterdam air cabinet operating table is ideal for producing hypothermia and for rewarming (Keuskamp 1961).

Equipment for extracorporeal circulation has been successfully used on animals inside the chamber. There are in fact very few techniques which cannot be carried out in such a large hyperbaric chamber.

A few practical points are worth mentioning, for the peace of mind of the anæsthetist. The cuff on the endotracheal tube is liable to leak if it is not 'topped up' during compression. During decompression it may burst or exert too much pressure on the trachea if some of the air in it is not released. Vaporizers should be kept open during compression and decompression. Transfusion apparatus should be checked as regards rate of the drip during compression and decompression. Long air inlet needles which extend above the fluid level in a transfusion bottle are safer than short ones. The gas flow passing through flow meters is reduced on compression and must be increased in relation to the increasing pressure. Gas cylinders therefore empty more quickly. Intrathoracic drains should not be clamped off before decompression is complete.

Carbon Dioxide Elimination
Haldane & Priestley (1935) have shown that when air is breathed at high pressures the partial pressure of carbon dioxide in the alveoli remains constant at 40 mmHg. The percentage CO_2 in the alveoli does vary, but it is the CO_2 tension which regulates breathing, and respiration is adjusted to keep the CO_2 tension at about 40 mmHg. During hyperbaric anæsthesia the production of CO_2 is not increased and CO_2 is not inhaled in appreciable amount (and not at all when 100% oxygen is administered). However, when oxygen is inhaled at 3 ATA there is sufficient oxygen dissolved in physical solution to render the hæmoglobin practically unnecessary for oxygen carriage and the hæmoglobin in venous blood remains 80–90% saturated. There is thus very little reduction of oxyhæmoglobin in the capillaries and the buffering effect of reduced hæmoglobin is lost. As a result there will be an increase in venous carbon dioxide tension. This in turn stimulates

hyperventilation so that a fall in arterial pCO_2 can be expected. Provided ventilation is controlled and adequate, there should be no difficulty in eliminating carbon dioxide during anæsthesia and this has been our experience in practice.

Nitrous Oxide
The problem of whether or not to use nitrous oxide as a light anæsthetic at 3 ATA is not so simple.

Very early experiments, in which curarized dogs were given the usual nitrous oxide with 30% oxygen for maintenance of anæsthesia at 3 ATA, resulted in death of the animals on decompression with formation of massive and alarming emboli of nitrous oxide in all the tissues. Nitrous oxide is 35 times more soluble in blood than nitrogen and the body tissues of an animal breathing the usual N_2O/O_2 percentages at 3 ATA soon absorb large quantities of nitrous oxide because of the increased partial pressure of N_2O at 3 ATA. On decompression to normal pressure, when the usual decompression table for air is followed, nitrous oxide will form bubbles in all the tissues where it was in solution.

For those who consider that nitrous oxide anæsthesia would be preferable to adding other agents, then the usual percentages of N_2O could be reduced compared with the percentages we used in our original experiments. Since the tension of N_2O required to produce anæsthesia is constant, the effective percentage at 3 ATA could be reduced to about one-third of that normally used. This would result in less N_2O dissolving in body tissues in unit time so that the danger of emboli would be reduced. This danger could probably be eliminated with reasonable safety if the nitrous oxide were washed out before decompression by the administration of oxygen.

The washout time of nitrous oxide with oxygen at 3 ATA could be calculated from the following data: percentage N_2O used, duration of administration, pulmonary blood flow, respiratory minute volume, mass of muscle, fat and water in the body and the solubility coefficients of N_2O in these various body tissues. According to the elimination curve of N_2O as given by Kety (1950) most of the gas would be eliminated within ten minutes, but it might not be completely eliminated for perhaps an hour. Therefore in patients with peripheral circulatory deficiency or shunts or respiratory depression the problem remains rather complicated and the results unreliable.

The Boston group are using 10% nitrous oxide in oxygen for operations upon infants with con-

genital heart disease. This would be equivalent to using 30% nitrous oxide at 1 ATA. However, it is probably too low a percentage to produce anæsthesia and in older children this would be a big disadvantage unless some other form of anæsthesia is added.   .

Another objection to the use of nitrous oxide is that, since the object of hyperbaric oxygen treatment is to obtain the highest possible oxygen tension, if nitrous oxide is used simultaneously then either the oxygen tension will be reduced by the presence of nitrous oxide or the working pressure used in the chamber will have to be further increased so as to maintain the required oxygen tension. Exposure to an increased working pressure would bring added risks to both operating team and patient. Another serious objection is that nitrous oxide combined with oxygen and halothane at 3 ATA produces an inflammable mixture.

Lastly, it is our policy to decompress as soon as the condition of the patient allows. Compression times for the team are thereby diminished. However, since the operation is not necessarily finished at this time, anæsthesia must be maintained; if we were using nitrous oxide as the sole anæsthetic, this would be very tricky because the percentage would have to be increased during decompression and decompression tables suitable for nitrous oxide would have to be followed. On the other hand, the operation could first be completed under N_2O anæsthesia at high pressure, but this would prolong the period of compression for surgical team and patient.

In view of these problems we decided that any advantage attached to the use of nitrous oxide is outweighed by the added risk.

Choice of Anæsthetic Agent
The choice of an anæsthetic agent under hyperbaric oxygen is limited to those which are non-inflammable within the pressure used. It should be effective at low concentrations in combination with 100% oxygen; give a smooth, controllable anæsthesia; allow rapid hypothermia. If high oxygen tensions are required, it should not cause too great a fall in cardiac output; the action of the agent on cerebral blood flow, combined with the concomitant change in oxygen requirements, should not produce an oxygen debt; monitoring of the EEG should be possible in order to detect early oxygen toxicity and to observe the depth of anæsthesia; provided pCO_2 levels are not allowed to rise unduly, it should not cause troublesome cardiac arrhythmias.

Our early choice fell on intravenous barbiturates and pethidine, but as soon as halothane became available, we used and have continued to use it for almost all our work, because it fits in with the requirements very well. Chloroform, trichloroethylene, methoxyflurane, neuroleptanalgesia (intravenous analgesic with butyrophenone derivative) could all be used. Like nitrous oxide, they do not appear to diminish cerebral bloodflows. Neuroleptanalgesia has the disadvantage that the patient does not sleep without nitrous oxide. Regional blocks combined with oxygen inhalation would seem to me to be ideal for some forms of surgery.

We started using a Fluotec vaporizer for halothane long before any calculations concerning the percentage halothane emitted at various settings under hyperbaric conditions had been worked out. We therefore monitored depth of anæsthesia by clinical observation, measurement of blood pressure and pulse and by the EEG changes.

Using high flows, high dial settings were never required. We have always paid special attention to humidifying the inspired gases.

Oxygen Toxicity
A danger which may affect the patient breathing pure oxygen is oxygen toxicity. It does not affect the surgical team whose members are breathing air at 3 ATA because the partial pressure of oxygen is only 60% of an atmosphere. Its exact ætiology is unknown and its onset is influenced by many factors. According to Behnke *et al.* (1935) a healthy, conscious person can tolerate inhaling 100% oxygen at 3 ATA for three hours without danger of oxygen intoxication. For reasons of safety, therefore, we have never drenched a patient with oxygen for longer than two hours at 3 ATA.

The lungs and the brain are the two organs mainly affected, but other organs such as the liver are also involved. If pure oxygen at normal pressure is inhaled then the *respiratory* effects are mostly seen such as œdema, thickening of the alveolar walls and pleuritis, even before symptoms occur. The animals become cyanotic and die. Sensitivity varies with species.

With high atmospheric oxygen convulsions develop which cause coma and death unless suitable counter-measures are instituted. The electrical pattern is similar to that of epileptic grand mal and the high voltage potentials are followed by electrical silence. Recovery, if it

occurs, is not usually complete for several hours. Further convulsions precede death. Under anæsthesia exposures can be longer before convulsions occur and lung damage is then more obvious. Barbiturates mask or prevent convulsions, but there is often more neurological damage afterwards.

There is a definite individual sensitivity and incidence varies with age, humidity, temperature, previous lung damage, toxicity and acid-base imbalance. The important fact is that anything increasing the arterial pCO_2 will cause convulsions to occur sooner, from which fact Bean concluded that oxygen intoxication was due to CO_2 intoxication. The administration of Tham buffer can prevent or retard the incidence of convulsions.

A conscious man breathing oxygen at either 1 ATA or at increased pressure has a reduction in cerebral blood flow resulting from cerebral vasoconstriction; however, under these conditions he hyperventilates because the venous pCO_2 increases: the arterial pCO_2 is then lowered. This may be a protective mechanism which tends to prevent brain tissue oxygen levels rising to values which might cause oxygen intoxication.

It is also an accepted fact that the higher the partial pressure of oxygen breathed, the sooner convulsions occur. They could therefore be precipitated by an increase in oxygen tension in the brain, occurring as a result of the cerebral vasodilatation, which follows an increase in arterial pCO_2. The anæsthetist should therefore take precautions to avoid high oxygen tensions in brain tissue, unless the condition of the patient calls for this, by hyperventilating.

High cerebral oxygen tensions can be avoided by hyperventilation, thereby reducing the arterial pCO_2 and causing a cerebral vasoconstriction and reduction in cerebral blood flow; oxygen tissue tension in other parts of the body would not be so appreciably reduced. Hyperventilation, by preventing the venous admixture and fall in arterial pO_2 known to be associated with anæsthesia (Bendixen *et al.* 1963), would also be beneficial in increasing the oxygen tension in the peripheral organs.

Conditions calling for hyperventilation would be peripheral traumatic vascular surgery and gas gangrene.

Increasing the oxygen tension in the brain is probably called for only if the oxygen available to the brain is diminished, as in cerebrovascular accidents and inadequate cerebral blood flow, and possibly in cerebral œdema following carbon monoxide hypoxia or circulatory arrest. In these conditions arterial pCO_2 should therefore not be actively reduced by hyperventilation. Ventilation should be adequate to prevent pulmonary shunting and a decrease in oxygen tension. In patients with arterial desaturation, as in right-to-left shunts, the high oxygen tensions liable to cause oxygen intoxication are not likely to occur at 3 ATA.

Monitoring of the EEG on an oscilloscope will give early evidence of an impending oxygen intoxication, seen as spiky potentials. It is particularly useful when the patient is anæsthetized and relaxed with muscle relaxants, as the electrical pattern can still be seen and appropriate action taken.

Other factors which influence the onset of oxygen toxicity, such as fever, metabolic acidosis and general toxicity, have been found to occur clinically in patients suffering from severe gas gangrene presented for hyperbaric oxygen therapy. It is beneficial to control the hyperpyrexia and correct the metabolic acidosis before treatment in order to reduce the liability to convulsions. In gas gangrene there may be generalized impaired circulation due to shock, and local impairment due to tissue damage and the clostridial gas production. It is therefore important to improve the local oxygen tissue tension, and simultaneously to try to prevent too high a rise of cerebral oxygen tension, by hyperventilation. Experimental work now being carried out in Amsterdam on conscious guinea-pigs seems to show that both respiratory and neurological signs of oxygen poisoning can be significantly reduced if the environmental humidity of the inspired oxygen is carefully controlled. These guinea-pigs all hyperventilate at 3 ATA oxygen.

Therapeutic Uses of Hyperbaric Oxygen
Hyperbaric oxygen has been used as therapy for various conditions, sometimes with excellent, sometimes with dubious, results. Comatose patients require the attention of an anæsthetist in the chamber.

It has definitely proved successful in abolishing rapidly the hypoxæmia due to carbon monoxide and opposing the secondary lack of oxygen in the cell, which develops after intoxication.

For gas gangrene it has been proved to be life-saving and limb-saving. The results of treatment of tetanus are not significantly better than those with intermittent positive pressure respiration.

The pain associated with peripheral ischæmic disease can be relieved for some days. Provided there is some blood supply, skin grafts of dubious viability could be tided over a crucial period until a better blood supply had developed – but our results are not yet significant.

Myocardial infarction treated in the caisson has not shown significant improvement but perhaps more heroic treatment is required.

Treatment of patients under hypothermia or hyperthermia together with hyperbaric oxygen for advanced inoperable tumours has reduced pain but has not significantly affected the tumours. I have no experience of the work of Churchill-Davidson et al. (1955) using X-ray therapy.

There is some experimental evidence that high pressure oxygen will alter the irreversible stage in hæmorrhagic shock and increase the survival rate.

Three patients in whom visual fields failed to improve on conventional therapy of anticoagulants and steroids improved rapidly on hyperbaric oxygen. All had some existing retinal blood flow. There was no improvement if the central retinal artery was completely blocked.

PROBLEMS ASSOCIATED WITH COMPRESSION
In patients who are comatose or anæsthetized the eardrums should always be perforated before compression. Patients who are awake and the medical staff inside the chamber are advised to swallow frequently and perform the Valsalva manœuvre in order to equalize pressures on both sides of the eardrums. Failure to equalize pressure results in pain and a tear in the drum may result. Should pain persist, in spite of the Valsalva manœuvre, in a medical attendant then he is advised to enter the lock and be decompressed. In patients who have pain and in whom treatment is essential the eardrums should be perforated. Attendants, who will be breathing air, not oxygen, should not be compressed if they are suffering from head colds, sinusitis or have dental cavities.

Decompression Sickness
Of great importance to the medical workers who are breathing air is caisson or decompression sickness or 'bends'. This is the reason that decompression is controlled from outside our chamber and to very strict decompression tables with pauses at 1·6 ATA and 1·3 ATA.

Nitrogen has a low solubility in blood but when air is breathed nitrogen is present at a very high concentration. Relatively a great deal dissolves in blood, especially at high pressures when the partial pressure is increased. Nitrogen will continue to dissolve slowly as it is taken up by the fatty tissues until they reach saturation. Therefore the longer the duration of the compression the longer decompression takes. If decompression occurs too rapidly, bubbles of nitrogen will be given off in the blood or tissues. Since caisson sickness is due to emboli of nitrogen, the patient who has been breathing oxygen or rich oxygen/air mixtures is far less apt to be the victim of 'bends', since oxygen is not an inert gas. Nitrogen is about six times more soluble in fat than in water so that it tends to be stored in fat tissue, therefore the obese are more prone to 'bends' than those who are thin.

Quite what the mechanism is, which causes 'bends', we do not know. Perhaps the nitrogen forms gas bubbles in the blood which are carried via the lung circulation into the general circulation or perhaps the bubbling takes place in the tissues.

Our own theory is that during decompression the removal of nitrogen which has accumulated in the fatty tissues is dependent on the circulation. Fatty tissue is not very vascular. Furthermore sitting on the gluteal region with its normally rather thick layer of fat will further minimize the circulation there. If the sitting position is maintained during decompression and the standing position is taken after decompression whereby circulation to the buttock improves, then nitrogen will be liberated as gas emboli. One surgeon and three anæsthetists who were rather obese and who remained sitting during decompression developed mild 'bends'. Symptoms disappeared on recompression and did not recur after a slower decompression. Active, but not excessive, movement is therefore advisable in all members of the team during decompression, in order to minimize the risk of 'bends'.

Bronchospasm, breath-holding or a blocked bronchiole may cause trapping of air in the lungs, which on decompression can cause pneumothorax, mediastinal emphysema, lung cysts, perhaps also emboli in the general circulation, and possibly syncope and death. We have not routinely inspired oxygen before starting and during decompression but it would increase the safety to the medical workers, provided it was not breathed too long. The fact that bone necrosis may present two or more years later as a result of 'bends' and even from symptom-free 'bends' is reason enough for taking maximal precautions and limiting compression times to a minimum.

Nitrogen Narcosis

Another danger associated with working in a hyperbaric chamber is the possible occurrence of nitrogen narcosis: the high tensions of nitrogen in blood which occur when air is inhaled at increased pressures, mainly above 6 ATA, have a narcotic action.

At 3 ATA, our working pressure, various tests were carried out in order to decide whether surgeons and anæsthetists breathing air would be adversely affected in their performances by the increased nitrogen tension in their blood. In some workers the first dive produced mild psychological changes such as a feeling of hilarity and loquacity but, after regular work in the chamber, this effect disappears. With regard to manual dexterity, there were no special difficulties associated with surgical or anæsthetic technical procedures. A study of handwritten surgical notes and anæsthetic charts made under 3 ATA pressure revealed no differences compared with those written at normal pressure. Handwriting and typewriting in air at 3 ATA during the course of four hours' exposure showed no significant changes. We conclude that air at 3 ATA for several hours has no detectable influence on psychological or manual performance in the majority of workers.

A new field for research, rather different from Boerema's original idea, has been opened up. Much is still unsolved and the situation can be compared with that surrounding hypothermia in the early 1950s. Nevertheless operations and therapies and anæsthesia using modern techniques are practical propositions in a hyperbaric chamber and many worth-while results have been achieved. Further research is necessary in order to minimize the risks and to clarify problems, particularly those associated with ventilation, blood flow and oxygen toxicity under high oxygen pressures.

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Observations on Hyperbaric Oxygenation during Anæsthesia

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The therapeutic use of hyperbaric oxygen rests on two assumptions: that significant increases in tissue oxygen tensions can be produced by inhaling oxygen at supra-atmospheric pressures and that such increases in oxygen tension can produce therapeutic benefit in ischæmic or hypoxic organs. Ideally, to test critically these basic assumptions, it would be necessary to measure tissue oxygen tension in each organ both in health and in disease. Since the interpretation of measurements made by tissue oxygen electrodes is difficult, it is reasonable to accept, for the present, measurements made on venous blood, cerebrospinal fluid or urine as giving some indication of what is happening at the cellular level.

The first link in the chain that ends in the tissues is the alveolar oxygen partial pressures achieved by breathing hyperbaric oxygen. Most large pressure chambers are pressurized with air and consequently oxygen has to be administered to the patient via some form of mask, hood or helmet. At first, in both Amsterdam and Glasgow, the BLB mask was the standard method of administering oxygen. The efficiency of this system of oxygen administration (as indicated by measurements of alveolar oxygen partial pressure) is shown in Table 1, which is based on results obtained in a study of 10 volunteers breathing oxygen at 2 atmospheres from a BLB mask at a flow rate of 8 litres/minute (McDowall, Ledingham, Jacobson & Norman 1965, in preparation). The average efficiency of the BLB mask was only 63% and there were wide variations in efficiency between individual subjects as indicated by the large standard deviation. These findings agree closely with those of Kory *et al.* (1962).

The performance of the BLB mask was therefore shown to be inadequate for the administration of oxygen in the pressure chamber. Following a suggestion by Squadron Leader J Ernsting

Table 1
Comparison of BLB mask and 'test' system of oxygen administration

	Mean alveolar oxygen partial pressure	Efficiency
BLB	898 ± 197 mmHg	63%
Test system	$1,253 \pm 108$ mmHg	88%